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Correlation of CT Cerebral Vascular Territories with Function: II. Posterior Cerebral Artery

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This paper presents schematic displays of the cerebral territories supplied by branches of the posterior cerebral artery as they would appear on axial and coronal computed tomographic (CT) scan sections. Companion diagrams of regional cortical function and a discussion of the fiber tracts are provided to simplify correlation of clinical deficits with coronal and axial CT abnormalities. Illustrations of the vascular supply and functional relay points (nuclei) of the thalamus are provided.

This report is the second in a series designed to correlate cerebral vascular territories and functional anatomy in a form directly applicable to computed tomography (CT) [1]. The illustrations are intended to simplify analysis of CT images in terms of clinical signs and symptoms and vascular territories in everyday practice.

Knowledge of vascular territories can help in differentiation between infarction and other pathologic processes. For example, if the position and extent of a lesion and the usual position and extent of a vascular territory are incongruous, infarction should receive relatively low diagnostic priority and vice versa. Knowledge of vascular territories can also facilitate correct interpretation of cerebral angiograms by pinpointing specific vessels for particularly close attention.

Knowledge of functional neuroanatomy applied to a patient's clinical features can improve detection of subtle lesions by pinpointing specific territories for special attention on CT and specific vessels for attention on angiograms.

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* The plane of coronal CT scans obtained depends on head positioning. If the coronal image is a reconstruction, the plane is perpendicular to the axial scans which may vary in their slope. Therefore, we have chosen to illustrate the vascular territories in the anatomic coronal plane. The reader must correct for minor differences in scanning angle. This will be facilitated by a review of coronal slices illustrated by Matsui and Hirano [2]

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Discussion

The vascular territories of the posterior cerebral artery and the neurologic functions ascribed to those territories are mapped on schematic axial and coronal* CT scans (figs. 1 and 2), and on maps of the medial surface of the cerebral hemisphere (fig. 3). The branches of the posterior cerebral artery have been divided into three groups: (1) the penetrating arteries to the brain stem, thalamus, and other deep structures, (2) the splenial branch to the corpus callosum, and (3) the branches to the cerebral hemisphere [3].

Penetrating Branches

Numerous very small branches arise from the proximal part of the posterior cerebral artery as it encircles the midbrain. These have arbitrarily been divided into one group that supplies the thalamus and hypothalamus and another that supplies the midbrain [3].

Thalamus and hypothalamus. The approximate origin and termination of the vessels that supply the thalamus are indicated in figure 4A. Each vessel in the drawing represents numerous very small branches. The pattern of arterial supply to the thalamus is variable; that shown in figure 4A is the most common, representing 30% of the brains studied by Schlesinger [3]. In general, four

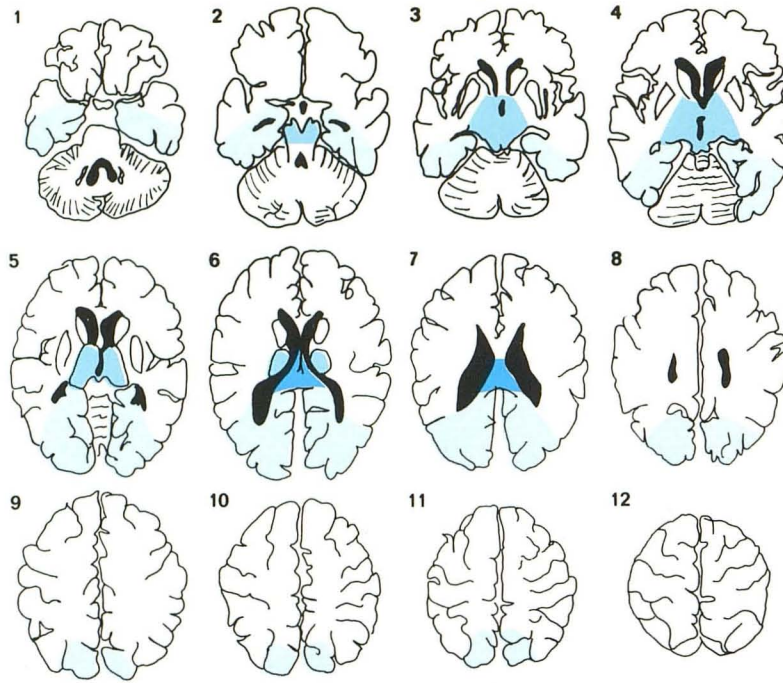


Fig. 1.—Axial CT scan diagrams arranged in sequence from base to vertex. Angle and levels of scan planes are shown in fig. 3. Territory of posterior cerebral artery is divided into three regions: thalamic and midbrain perforators (medium blue) callosal (dark blue) and hemispheric (light blue).

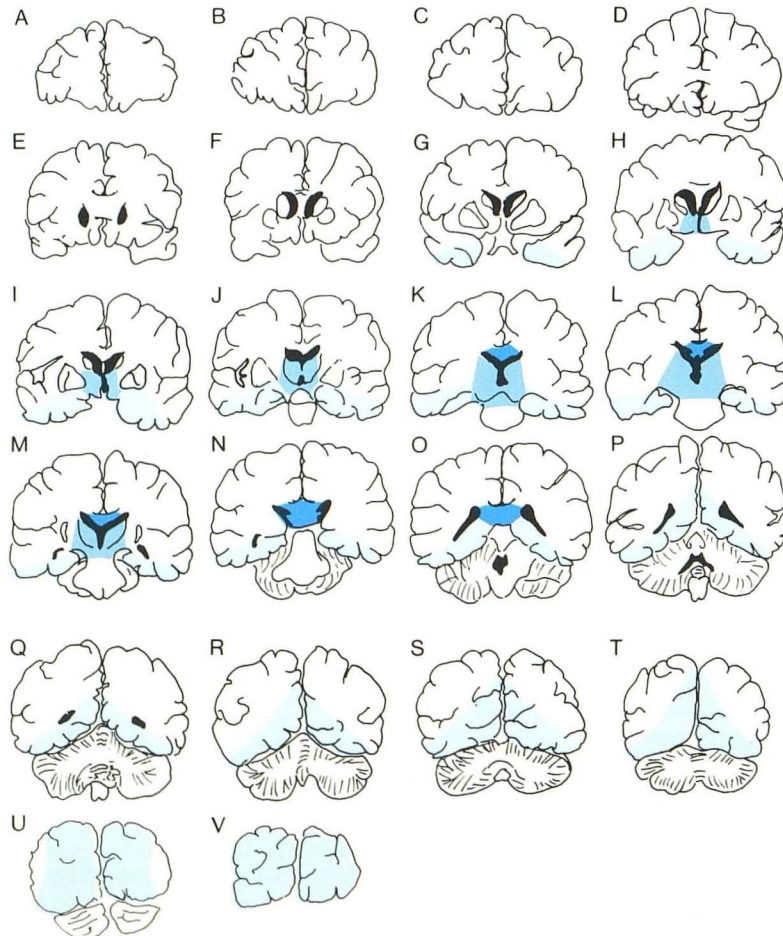


Fig. 2.—Coronal CT scan diagrams arranged in sequence from front to back. Angle and levels of scan planes are shown in figure 3. Territories of posterior cerebral artery are identified as in fig. 1.

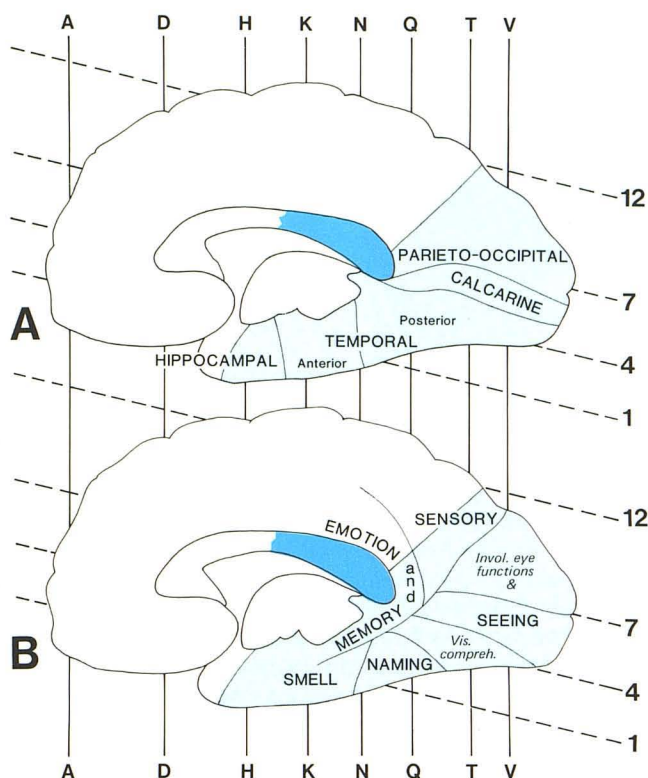


Fig. 3.—**A**, Regions supplied by the five hemispheric branches of posterior cerebral artery localized on medial view of right cerebral hemisphere. **B**, Functional cortical regions supplied by posterior cerebral artery localized on medial view of right cerebral hemisphere. See axial and coronal scan levels in figs. 1 and 2 for correlation.

groups of perforating vessels supply the thalamus: (a) premamillary, (b) thalamoperforators, (c) thalamogeniculate, and (d) posterior choroidal and cingulate branches.

As illustrated in figure 4A, the *premamillary arterial group* (a) supplies the anterior thalamus (the anterior, medial, ventral anterior, and ventral lateral nuclei). The anterior nucleus connects with the mamillary bodies and the hippocampus and is part of the memory and emotion circuit of Papez [4, 5]. The dorsal medial nucleus connects with the frontal lobe, the amygdala, and possibly the hypothalamus. Damage to either of these nuclei can cause profound memory loss and personality change (fig. 4B). Usually the recent memory is most severely affected (Wernicke-Korsakoff syndrome) [6]. The specific anatomy and physiology underlying these effects is a subject of controversy and was reviewed recently by Horel [7, 8]. The ventral anterior and lateral nuclei influence the motor system, particularly the extrapyramidal and cerebellar systems; their ablation may relieve some symptoms of Parkinson disease [9]. The lateral nucleus helps coordinate motor and sensory impulses but has no clinically defined function (see figs. 4B and 5).

In addition to the thalamus, the premamillary arteries supply the intermediate part of the hypothalamus, an area that forms the central one-third of the walls of the third ventricle, and the mamillary bodies. There is collateral supply to this area from internal carotid branches [10]. This part

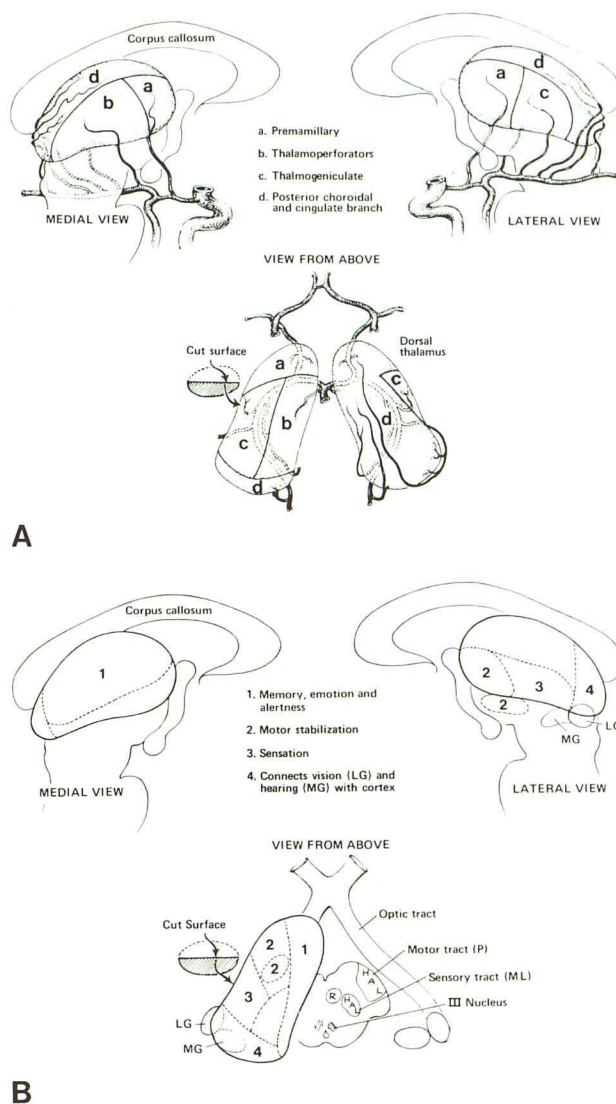


Fig. 4.—**A**, Schematic view of four vascular territories of thalamus. Each is represented by single arterial branch at approximate origin and termination of numerous small perforating branches: (a) premamillary, (b) thalamoperforator, (c) thalamogeniculate, and (d) posterior choroidal and cingulate branches. This pattern represents 30% of brains [3]. Midbrain perforators (the largest of which is the quadrigeminal plate artery) are *not* illustrated. **B**, Four functional relay areas of thalamus: (1) memory and emotion (anterior and dorsal medial nuclei), (2) motor stabilization (ventral anterior, ventral lateral, and subthalamic nuclei), (3) sensation (ventroposteromedial and ventroposterolateral nuclei), and (4) connections of visual (LG = lateral geniculate) and hearing (MG = medial geniculate) areas with the parietal cortex via the pulvinar. Unmarked areas are nonspecific association areas which are poorly understood. Lesions in area (1) result in neuropsychologic symptoms; in area (2) they result in hemiballismus (or curb Parkinson disease contralaterally); in area (3) they result in Dejerine-Roussy syndrome; and in area (4) they cause aphasia, if the left thalamus is affected. Midbrain sectioned at level *below* inferior colliculi shows major midbrain motor tract (P = pyramidal) and sensory tract (ML = medial lemniscus) divided into areas for head (H), arm (A), and leg (L) fibers. These tracts control side of body opposite lesion. Red nucleus (R) is pathway for fibers from cerebellum to thalamus. Lesions here affect coordination contralaterally.

of the hypothalamus is the source of somatostatin [11] (which blocks the release of growth hormone and thyroid stimulating hormone) and thyroid releasing hormone [12]. Neuropathways carrying vasopressin and oxytocin to the posterior pituitary gland as well as pathways for leuteinizing

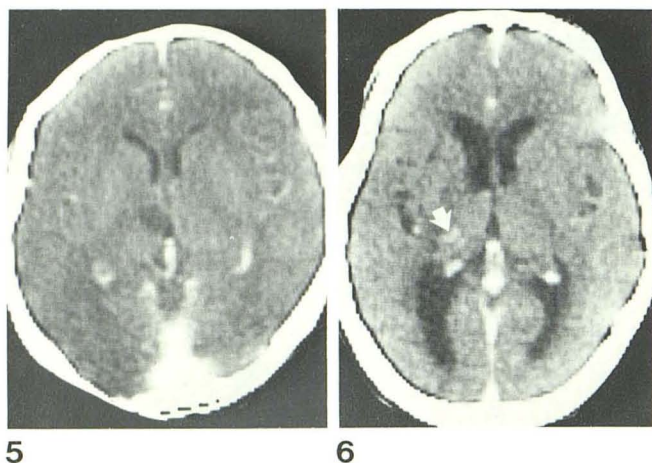


Fig. 5.—Postinfusion axial CT scan (at scan level 4 of fig. 1). Infarction of anterior aspect of right thalamus, which is usually supplied by premamillary branches of posterior communicating artery (area a in fig. 4A). Patient had sudden onset of severely diminished mental ability and motivation (area 1 in fig. 4B). Psychiatric evaluation suggested organic cause for disability, prompting CT evaluation.

Fig. 6.—Axial CT scan (at level 5–6 in fig. 1). Enhancing infarction (arrow) in thalamogeniculate territory (area c in fig. 4A). Patient had Dejerine-Roussy (thalamic) syndrome of altered sensation (area 3 in fig. 4B).

hormone releasing factor, corticotrophic releasing factor, and prolactin inhibitory factor [12] travel through this region. Therefore, intermediate hypothalamic lesions can cause a complete loss of endocrine function, simulating total section of the pituitary stalk. Lesions in this area cause hyperphasia, anorexia, and rage attacks [11].

The premamillary artery together with branches from the thalamoperforating arteries also supplies the posterior part of the hypothalamus [10]. Tumors in this area produce precocious puberty [11], disturbances of temperature regulation [13], and disturbances of consciousness with somnolence [11]. (The anterior hypothalamus forms the anterior walls of the third ventricular chamber and influences temperature regulation, fluid electrolyte balance, synchronization of sleep and wakefulness, and perhaps some aspects of cardiac rhythm. It is supplied by small perforating branches from the anterior cerebral artery; infarctions in this area can cause abnormality in any of its functions.)

As illustrated in figure 4A, the *thalamoperforating arterial group (b)* supplies the medial ventral part of the thalamus (centromedianum and parafascicular nuclei). This territory mediates many aspects of arousal, attention, and alertness [4]. Thalamoperforating branches derived from one posterior cerebral artery commonly supply all or part of the contralateral territory as well as the ipsilateral territory [14].

As illustrated in Figure 4A, the *thalamogeniculate group (c)* supplies the lateral ventral part of the thalamus (pulvinar, ventroposterior lateral, and ventroposterior medial nuclei). The pulvinar may coordinate visual and auditory information by receiving input from the lateral and medial geniculate bodies and projecting to the occipital and temporal lobes [4]. Lesions of the left pulvinar area can produce language dysfunction (aphasia or dysphasia) [15]. The ventral poste-

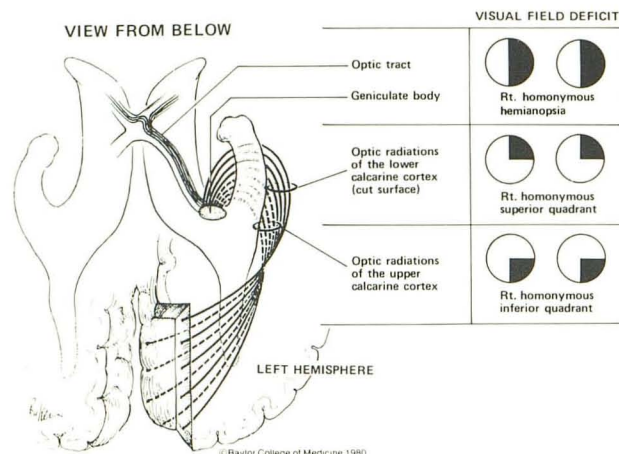


Fig. 7.—Visual pathways viewed from below. Visual defects produced by lesions along left optic tract. Radiations are very close to ventricular walls.

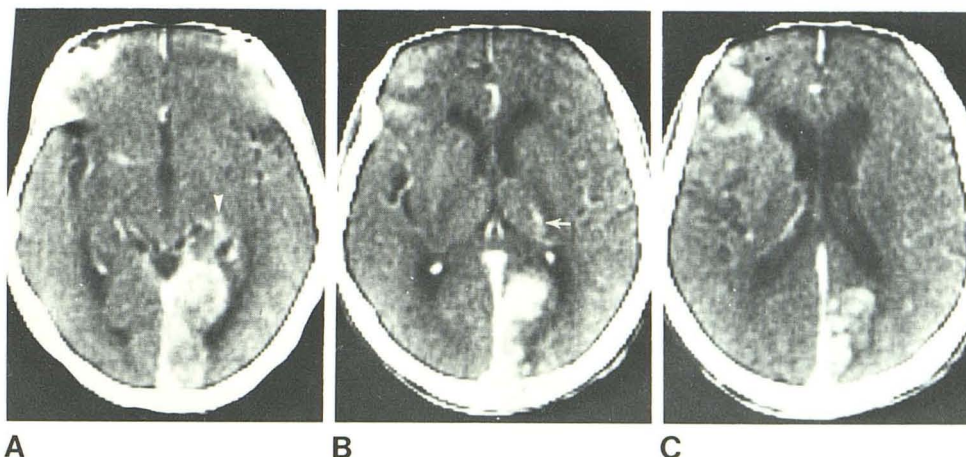
rior lateral and ventral posterior medial nuclei relay sensory information from body and face, respectively [4]. Lesions produce an initial insensitivity to pain that evolves over a period of months into spontaneous or inappropriately elicited pain in the affected body part. This has been named *anesthesia dolorosa* and is found in the Dejerine-Roussy (thalamic) syndrome [16] (see figs. 4 and 6).

The thalamogeniculate branches also supply the lateral geniculate body, the medial geniculate body, and structures below the anterior and medial part of the thalamus (e.g., Forel fields in the subthalamic area). Lesions of the lateral geniculate body cause loss of sight in the half of the visual field opposite the lesion [4] (see figs. 7 and 8). The medial geniculate body is part of the hearing pathway but unilateral lesions of the medial geniculate body do not cause clinical hearing problems since there is bilateral representation of hearing at this level due to previous decussation. Lesions in the vicinity of the subthalamic area and Forel fields can cause violent flinging movements of the opposite extremities called *hemiballismus* as well as the uncontrollable jerking and writhing movements of *choreoathetosis* [17, 18]. However, only *hemiballismus* has good localizing value and suggests a lesion in the nucleus subthalamicus. Since the subthalamic region receives collateral supply from the anterior choroidal artery, these manifestations are rare in infarction but common in hemorrhage [18].

As shown in Figure 4A, the *choroidal and cingulate branches (d)* supply the posterior and superior thalamus (anterior, medial, pulvinar, and habenular nuclei). There are extensive anastomoses in these areas [14, 19] and infarction is rare. Symptoms resulting from lesions in the first three of these thalamic nuclei have been described in the previous discussion. There is no known specific clinical deficit produced by habenular lesions per se. In addition to the thalamus, the posterior choroidal branches supply the pineal, choroid plexus, crus, commissure, and body of the fornix, part of the anterior columns of the fornix, and part of the lateral geniculate body [14].

Midbrain. The second group of small penetrating

Fig. 8.—Axial contrast CT scans (levels 3–7, fig. 1). Enhancing infarction of cortex supplied by left posterior temporal, calcarine, and parietooccipital arteries, and thalamogeniculate territory (arrow), which includes medial part of lateral geniculate (arrowhead). This produced loss of entire right half visual field (contralateral homonymous hemianopsia (see fig. 7). Sensory changes were nominal. Also, note infarction in distribution of prefrontal branch of right middle cerebral artery.



branches supplies the midbrain [19]. They enter the midbrain substance perpendicularly, either directly at their point of origin from the posterior cerebral artery or after accompanying the parent artery around the midbrain for some distance [19, 20]. Others term these "thalamoperforators" and the syndromes produced by their occlusion "thalamoperforate syndromes" [21]. Since this group of vessels does not supply the thalamus it would be better to call them "midbrain perforators" and the associated syndromes "midbrain perforate syndromes."

The midbrain contains many vital pathways and nuclei; lesions here have a variety of clinical manifestations. Some of these deficits have eponyms (e.g., Claude, Nothnagel, Weber, and Benedikt syndromes, among others). There is disagreement in the literature as to the precise constellation of symptoms found in each of these syndromes, so these eponyms will not be used in our discussion [19, 21, 22]. Only a *combination* of deficits can be used to localize a lesion to the brainstem, because an isolated lesion in structures outside the midbrain can produce identical deficits. It is the combination of deficits that enables localization of a lesion to the midbrain. Lesions within or pressing on the midbrain produce identical symptoms by compromising one or more of the vital centers in the midbrain. Therefore, the etiology cannot be determined by clinical means.

Damage to parts of the midbrain can lead to: (1) third nerve paralysis causing symptoms in both eyes but most marked in the eye on the same side as the lesion; (2) disturbed sensation, strength, and/or coordination on the side opposite the lesion; (3) depression of consciousness; and (4) decerebrate rigidity. A detailed description of these deficits follows. The reader will find it helpful to refer to figure 4B to visualize the position of the third nerve nucleus, red nucleus, and motor and sensory tracts in the midbrain as these structures are discussed.

A total *third nerve paralysis* results in ptosis, a dilated unreactive pupil and downward and outward deviation of the eye ipsilaterally. Injury to peripheral third nerve fibers (after they leave the brain stem or as they travel through the cisterns to reach the eye) causes some or all of these symptoms ipsilaterally. Brain stem lesions may cause third nerve symptoms bilaterally since the nuclei are close to-

gether. Isolated brain stem lesions involving a single third nerve nucleus are therefore very rare (see fig. 4B). Brain stem lesions may simulate peripheral third nerve lesions by compromising nerve fibers in the brain stem before they exit. Some authors have stated that third nerve paralysis without pupillary abnormality indicates infarction rather than compression by a mass, but this distinction is unreliable [23–26].

There are fibers of the third nerve at the collicular level of the midbrain that can be damaged by tumor compression (e.g., pinealoma) producing Parinaud syndrome, [26, 27]. This consists of loss of upward gaze, impaired pupillary reactivity, and convergence nystagmus, which is not true nystagmus, but, rather, a simultaneous contraction of all the extraocular muscles. Convergence results because the medial recti are the strongest muscles of the group [26].

Disturbances of sensation or movement are caused by damage to the major sensory tract (medial lemniscus) or motor tract (pyramidal) (see figure 4B). Lesions here cause contralateral hemianesthesia and/or hemiparesis and may affect the head (H), arm (A), or leg (L) in varying degrees [28].

Damage to the red nucleus, which coordinates cerebellar thalamic fiber tracts (see fig. 4B), results in impaired coordination and control of movement contralaterally [29]. There is tremor during movement (perpendicular to the direction of motion) that worsens as the target is approached. One also finds dysmetria, difficulty with rapid alternating movements and inability to check or stop a previously initiated movement [27, 30, 31]. Another related type of tremor, rubral tremor, may also be seen. This tremor is present at rest, although it increases during movement. It involves the limb more proximally than does the usual cerebellar tremor. A third type of tremor, seen only at rest, which is similar to that seen in Parkinson disease, may also be encountered [27, 30, 31]. In addition to the tremor, there may be contralateral hemiballismus and choreoathetosis (as described previously) if there is damage to the adjacent substantia nigra and/or subthalamic region [21, 29, 30, 32].

Depression of consciousness is presumably caused by damage to the central or paramedian reticular midbrain

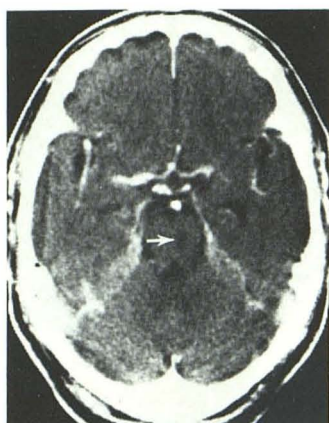


Fig. 9.—Axial CT scan (level 2, fig. 1). Left midbrain infarction (arrow). Patient had right hemiparesis with facial sparing and dysarthria and left third nerve palsy, later resolved, but right-sided sensation remained impaired. There is only a partial correlation between actual deficits and those based on fig. 4B.

activating system. It can be also caused by destruction or depression of the reticular system in the pons or medulla or by bilateral hemispheric dysfunction. This impairment of consciousness is of little localizing value [27].

Decerebrate rigidity can be produced in animals by lesions in the regulating structures between the quadrigeminal plate and the lateral vestibular nucleus in the pons [27, 33]. The subject assumes a position in which the legs are rigidly extended and the arms are extended, adducted, and hyperpronated [25]. This posture is not as reliable a localizing indicator in humans as it is in other animals [23].

In our experience, CT of midbrain infarctions has not correlated well with the clinical presentations. This may be due to brain stem damage not demonstrated by CT (see figs. 4B and 9).

Callosal Branches

The callosal arteries are a plexus of small vessels that arise from the parietooccipital or lateral choroidal branches and penetrate the upper surface of the posterior one-half of the corpus callosum. The plexus may be replaced by a single branch [14]. Infarction can result in separation of the language-dominant left hemisphere from the right hemisphere, which mediates function of the left side of the body. If the patient's left occipital lobe and splenium are infarcted, he can speak, write, and understand speech but he becomes unable to read with his remaining left visual field despite the fact that he can see the letters. (He cannot transfer the visual images received by the right occipital lobe for interpretation in the left hemisphere.) This is called alexia without agraphia. It is rare and may be partial [34].

Hemispheric Branches

The posterior cerebral artery has five cortical branches. The patterns of infarction can be simplified by comparing the vascular territories (fig. 3A) with the corresponding functional areas (fig. 3B). Infarction of cortical branches of the posterior cerebral artery is responsible for several inter-

esting clinical syndromes. The calcarine artery (sometimes together with the parietooccipital artery) supplies the calcarine cortex on one side. Infarction thereof causes homonymous hemianopsia of the contralateral visual fields (fig. 8). It is commonly stated that, with lesions of the calcarine cortex, the central macular part of the visual field is spared due to collateral circulation [35]. We have rarely seen this.

Infarction of the calcarine cortex bilaterally results in complete blindness. However, there is sometimes also an unusual constellation of symptoms known as Anton syndrome in which the patient, although blind, is unaware of the blindness and denies it [36]. Sometimes he will construct elaborate descriptions of his surroundings. If allowed he will often walk into walls or trip over objects. Patients sometimes also display difficulty in tactile identification and enumeration of objects. Apraxia of ocular movements may also be encountered. It is in bilateral occipital infarction that macular sparing is most commonly reported. Macular sparing in these instances may actually represent partial recovery from total blindness [34]. Damage to the calcarine cortex can also produce unformed visual hallucination, metamorphosia (distortion of shape), teleopsia (an illusion in which near objects appear to be at a great distance), distortion of visual outlines, and other aberrations of visual perception [36].

Damage to the nondominant calcarine cortex as well as damage to the nondominant lingual gyrus, which are supplied by the calcarine and temporal branches (fig. 3B), may produce topographic disorientation and prosopagnosia. Prosopagnosia is the inability to recognize familiar faces even though they can be clearly seen [36].

Damage to the cortex above and below the calcarine fissure, particularly in the dominant hemisphere, destroys the visual association areas that control involuntary eye function and visual comprehension (see fig. 3B). These areas are supplied by the parietooccipital and posterior temporal branches of the posterior cerebral artery. Patients with lesions in these areas rarely may continue for several seconds to see the image of an object that is quickly removed from view. They may also not perceive an object in the damaged visual field if another identical object is simultaneously presented in the normal visual field. This phenomenon is called extinction [36].

The anterior temporal branch supplies only the inferior aspect of the anterior temporal lobe; the tip of the temporal lobe is supplied by the middle cerebral. The cortex in this area is involved in naming objects.

The hippocampal branches of the posterior cerebral artery supply the hippocampal formation including its projections, the fornices, and the psalterium (fig. 3B). The most commonly observed clinical finding in patients with bilateral hippocampal infarcts is memory deficit. This may also be seen with unilateral deficits on the dominant side. Lesions in this area may also impair the sense of smell [36].

The functional neuroanatomy and vascular territories supplied by the posterior cerebral artery have been graphically summarized in a format directly applicable to CT to simplify correlation of CT images, clinical signs and symptoms, and vascular territories in everyday practice. We hope this infor-

mation will enhance the usefulness and accuracy of CT in evaluating cerebral lesions.

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